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Issue Date: 04 October 2005

In the Matter of

Marjorie M. Roland, Widow of
Boyd M. Roland,
Claimant
v.

Eastern Associated Coal Corp.,
Employer

and

Director, Office of Workers' Compensation Programs,
Party-In-Interest

Case No. 2004BLA06016

DECISION AND ORDER
AWARDING BENEFITS

This proceeding arises from a claim for benefits filed by Marjorie M. Roland, the surviving spouse of Boyd M. Roland, a deceased coal miner, under the Black Lung Benefits Act, 30 U.S.C. §901, et seq. Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Federal Regulations.[1]

Black lung benefits are awarded to coal miners who are totally disabled by pneumoconiosis caused by inhalation of harmful dust in the course of coal mine employment and to the surviving dependents of coal miners whose death was caused by pneumoconiosis. Coal workers' pneumoconiosis is commonly known as black lung disease.

A formal hearing was held before the undersigned on February 15, 2005 in Abingdon, Virginia. At that time, all parties were afforded full opportunity to present

[1] The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 Wednesday, December 20, 2000. The revised Part 718 regulations became effective on January 19, 2001. Since the current claim was filed on January 31, 2002 (DX 4), the new regulations are applicable.

evidence and argument as provided in the Act and the regulations. Claimant appeared at the formal hearing, and Claimant and Employer were represented by their respective counsel.

The record consists of the hearing transcript, Director's Exhibits 1 through 54 (DX 1-54), Claimant's Exhibits 1 through 9 (CX 1 - 9), and Employer's Exhibits 1 through 19 (EX 1 - 19). The Claimant filed her brief on September 1, 2005. Although the Employer twice requested an extension of time to file a brief, the Employer did not file one.

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the deposition testimony presented.

Procedural History

On January 9, 2002, Mr. Roland, a former coal miner, passed away (DX 10). On January 31, 2002, the Claimant, Marjorie M. Roland, filed the current application for Black Lung benefits under the Act, as his surviving spouse (DX 4).[2] On October 16, 2003, the District Director (Director) issued a Proposed Decision and Order awarding benefits (DX 46). Following the Employer's timely request for a formal hearing (DX 48), this matter was referred to the Office of Administrative Law Judges for *de novo* adjudication. As stated above, a formal hearing was held before the undersigned on February 15, 2005, and the record was held open for the submission of additional evidence, as well as post-hearing briefs.

Issues

The issues contested by the Employer are:

1. Whether Mr. Roland was a miner.
2. Whether Mr. Roland worked as a miner after December 31, 1969.
3. The length of Mr. Roland's coal mine employment.
4. Whether Mr. Roland had pneumoconiosis.
5. If so, whether his pneumoconiosis arose out of his coal mine employment
6. Whether Mr. Roland's death was due to pneumoconiosis.

(DX 53; Tr. 27-28).

Applicable Standard

The Regulations at 20 C.F.R. § 718 apply to survivors' claims which are filed on or after April 1, 1982. 20 C.F.R. § 718.1. Because Claimant filed her survivor's claim after January 1, 1982, 20 C.F.R. § 718.205 applies to this claim.

[2] Mr. Roland filed a claim for benefits in 1982, and after the Employer agreed to pay benefits, the Director issued a Decision awarding benefits on May 3, 1984 (DX 2).

The regulations provide that a survivor is entitled to benefits only where the miner died due to pneumoconiosis. 20 C.F.R. § 718.205(a). The Claimant must establish that: (1) the decedent was a coal miner; (2) the decedent suffered from pneumoconiosis at the time of his death; (3) the decedent's pneumoconiosis arose out of his coal mine employment; and (4) the decedent's death was caused by pneumoconiosis or pneumoconiosis was a substantially contributing cause or factor leading to his death. All elements must be established by a preponderance of the evidence. *Strike v. Director, OWCP,* 817 F.2d 395, 399 (7th Cir. 1987). The survivor of a miner who was totally disabled due to pneumoconiosis at the time of death, but died due to an unrelated cause, is not entitled to benefits. 20 C.F.R. § 718.205 (c). If the principal cause of death is a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 C.F.R. § 718.205 (c)(4).

The Board has held that death will be considered to be due to pneumoconiosis where the cause of death is significantly related to or significantly aggravated by pneumoconiosis. *Foreman v. Peabody Coal Co.*, 8 B.L.R. 1-371 (1985). The United States Court of Appeals for the Fourth Circuit, in which the instant case arises, has held that pneumoconiosis is a substantially contributing cause of death if it hastens, even briefly, the miner's death. *See, Shuff v. Cedar Coal Co.,* 967 F.2d 977 (4th Cir. 1992), *cert. denied,* 113 S.Ct. 969 (1993). *See also, Brown v. Rock Creek Mining Corp.,* 996 F.2d 812 (6th Cir. 1993)(J. Batchelder dissenting); *Peabody Coal Co. V. Director, OWCP,* 972 F.2d 178 (7th Cir. 1992); *Lukosevicz v. Director, OWCP,* 888 F.2d 1001 (3rd. Cir. 1989).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Length of Coal Mine Employment/Responsible Operator

The Director determined that Mr. Roland worked as a coal miner for 32 years. (DX 46). This is supported by Mr. Roland's Social Security Earnings record, as well as records from Eastern Associated Coal (DX 5-7). Indeed, a letter from Eastern Coal states that Mr. Roland worked for Eastern from March 1952 to June 1982, when he retired (DX 5, 6). I find that Mr. Roland worked as a coal miner after December 1969, and that he had at least 32 years of coal mine employment. Additionally, I find that the Employer is properly designated as the responsible operator.

Personal History/Eligible Survivor

Mr. Roland was born on June 1, 1925. He married his wife, Marjorie (nee Murray), on August 15, 1946 (DX 9). They remained married until Mr. Roland's death on January 9, 2002. Accordingly, I find that the Claimant is an eligible survivor of the miner. The Claimant has no dependents for purposes of augmentation of benefits.

Medical Evidence

The case file contains various chest x-ray readings, arterial blood gas results, medical records, and physicians' opinions, which were submitted in conjunction with the survivor's claim, as summarized below.

X-Ray Evidence

X-Rays Performed During Hospitalizations

Mr. Roland underwent numerous x-rays and CT scans during hospitalizations in his last years. The narrative findings are as follows.

Dr. David L. Groten administered an x-ray on October 6, 1999 (DX 21). He noted bilateral pulmonary infiltrates and/or masses, as seen in a previous study of October 4, 1999. The findings were slightly more prominent in the right mid lung region. In reviewing an x-ray performed on October 4, 1999, Dr. Groten noted a patchy increased density in the right perihilar region and left mid lung (DX 22). He also identified a slightly increased density at both lung bases, which could represent bilateral pneumonia. He felt that the possibility of bilateral pulmonary masses could not be excluded.

Dr. A. K. Raja Rao reviewed an x-ray performed on March 17, 2000 (DX 23). He found evidence of complicated pneumoconiosis, in the form of conglomerate perihilar densities, retraction, and some pleural thickening on the right side. He stated that a superimposed right moderate pleural effusion was present, with a suggestion of a few septal lines at the base. According to Dr. Rao, the findings could be due to congestive heart failure. His conclusion was severe complicated pneumoconiosis, and a small right pleural effusion, possibly due to congestive heart failure.

Dr. Shahan reviewed an x-ray performed on March 20, 2000 (DX 19, 24). He indicated that it showed severe chronic reticulonodular interstitial disease, with bilateral perihilar conglomerate masses. The right chest had extensive chronic pleural thickening, findings unchanged from March 17, 2000 and October 4, 1999. His impression was chronic severe interstitial disease.

Mr. Roland underwent an x-ray on September 1, 2000, which was reviewed by Dr. Rao (DX 25). He noted that there were bilateral perihilar opacities, compatible with conglomerate masses related to complicated pneumoconiosis. Dr. Rao also stated that there was pleural thickening at the right lateral chest and base. The right sided perihilar opacity and pleural densities were more prominent on this x-ray, as compared to the March 20, 2000 x-ray. His conclusion was complicated pneumoconiosis and chronic right sided pleural densities.

Dr. Thomas E. Miller reviewed frontal and lateral views of a December 20, 2000 x-ray (DX 26). He noted findings consistent with severe complicated pneumoconiosis, with large bilateral conglomerate masses and right pleural thickening. His impression

was that the findings were consistent with COPD and severe complicated pneumoconiosis. Dr. M. Rahman reviewed a portable x-ray of the same date, noting bilateral conglomerate perihilar opacities consistent with complicated pneumoconiosis, and progressive massive fibrosis. He also noted diffuse nodular interstitial densities consistent with interstitial fibrosis (DX 27). His impression was COPD, and severe complicated pneumoconiosis and progressive massive fibrosis.

Mr. Roland underwent a CT scan on February 26, 2001 (DX 15, 28). At that time, he had severe shortness of breath, and his physicians suspected that he could have a pulmonary embolus. Dr. Cappiello reviewed the CT scan of his thorax, which showed no evidence of pulmonary embolism, but did show multiple conglomerate masses of pneumoconiosis, a large right pleural effusion, and a small 2 cm. thin walled cavity with air fluid in the superior segment of the left lower lobe, which he felt could represent a conglomerate mass that had undergone necrosis and spontaneously drained. Dr. Cappiello noted a thin walled cavity with an air fluid level in the superior segment of the left lower lobe, and large conglomerate masses in the left and right perihilar regions, mainly in the upper lobes, with the typical appearance of a conglomerate mass of progressive massive fibrosis.

Dr. Cappiello administered a chest x-ray on May 24, 2001, which showed a conglomerate mass that had undergone necrosis and spontaneously drained. He found advanced complicated pneumoconiosis, with large bilateral conglomerate masses, unchanged from a previous study. There was also hyperinflation of the lungs, with changes of underlying COPD. There was extensive pleural scarring in the lateral and posterior chest walls. Dr. Cappiello's impression was advanced complicated pneumoconiosis (DX 16, 29).

Dr. D. Fowler reviewed a June 1, 2001 x-ray, noting prominent changes of complicated pneumoconiosis, with conglomerate masses in the right and left mid lung, and interstitial fibrosis scattered in both lungs (DX 30).

Dr. Miller reviewed Mr. Roland's December 29, 2001 x-ray, finding severe complicated pneumoconiosis, with large conglomerate masses bilaterally (DX 31).

Dr. E. Aycoth reviewed x-rays performed on January 6, 2002 (DX 32). On the left lateral view, he noted a pleural reaction, with no evidence of layering left pleural effusion. There was progressive massive fibrosis, and conglomerate lung masses of complicated pneumoconiosis. On the right view, he noted moderate layering right pleural effusion, and a conglomerate right lung mass or progressive massive fibrosis.

Dr. Michael Alexander

Dr. Michael Alexander reviewed a series of chest x-rays and CT scans from 1999 through 2001, and prepared a report dated December 23, 2004 (CX 5).[3] On his review of

[3] Dr. Alexander's report is technically outside the evidentiary limitations of the new regulations because it relies on inadmissible evidence. *See* 20 C.F.R. § 725.414(a)(2), (a)(3), *Dempsey v. Sewell Coal Co.*, 23

the March 25, 1999 x-ray, Dr. Alexander noted the presence of small round opacities bilaterally, consistent with pneumoconiosis 2/2, q, r. There was an area of coalescence in the right upper zone, and bilateral large opacities of complicated pneumoconiosis. The largest mass extended from the right upper zone to the right hilum, and measured approximately 8 x 6 cm. There were at least two large opacities in the left lung, with the upper zone opacity measuring 3.5 x 1.5 cm., and the mid zone measuring 3 x 2.5 cm., and partially calcified. According to Dr. Alexander, these are classified as category B complicated pneumoconiosis.

On Mr. Roland's March 20, 2000 x-ray, Dr. Alexander noted the same background of small opacities, and coalescence in the right upper zone. As well, there were bilateral large opacities of complicated pneumoconiosis. The conglomerate mass in the left mid zone was more clearly defined, and actually appeared to measure 7 x 3.5 cm. He stated that these represented category B complicated pneumoconiosis. Additionally, he felt that it was reasonable to argue that the combined area of the large opacities met the criteria for classification as category C.

Dr. Alexander felt that Mr. Roland's December 20, 2000 x-ray showed no significant change, except for the suggestion of a possible 2 cm. nodule or large opacity in the left lower zone. The May 24, 2001 x-ray was similarly unchanged. Although the June 1, 2001 x-ray did not show significant change, it did suggest a left lower zone large opacity.

Dr. Alexander also reviewed Mr. Roland's February 26, 2001 CT scan, noting two large opacities in the right lung, one measuring 4.5 x 4 cm., and one 3 cm. in greatest diameter. On the left, there was one contiguous large opacity, extending from the upper zone to the mid zone, with a vertical length of 7 cm., and a width of 5 cm. All of the masses were similar in appearance, with smooth non-spiculated well-defined margins, central low attenuation areas, and internal calcifications. According to Dr. Alexander, the location and appearance of these masses was completely characteristic of the conglomerate fibrosis seen in complicated pneumoconiosis. He also noted a similar mass in the left lower zone, measuring 2 cm. in diameter, and containing an air/fluid level, implying central cavitation, which can occur in conglomerate masses of pneumoconiosis.

Mr. Roland's December 23, 2000 CT scan showed no change in the bilateral large pulmonary masses, which Dr. Alexander characterized as completely characteristic of the

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B.L.R. 1-47 (2004) (en banc). The x-rays relied on by Dr. Alexander exceed Claimant's evidentiary limitations. The regulations dictate "A medical report may be prepared by a physician who examined the miner and/or reviewed the available admissible evidence." 20 C.F.R. § 725.414 (a)(1). The Board has interpreted this regulation and found that medical data underlying a medical report must itself be admissible. *Dempsey v. Sewell Coal Co.*, 23 B.L.R. 1-47 (2004) (en banc). However, the regulations provide that I may admit evidence that exceeds the limitations upon a finding of good cause. At the hearing, after a discussion with the parties, I admitted Dr. Alexander's report, as well as the x-rays on which he relied, based upon my finding that, in a case such as this that involves a question of the existence of complicated pneumoconiosis, it is important to review longitudinal and serial x-ray evidence. The Employer was also provided the opportunity to have Dr. Alexander's report, as well as the x-rays on which he relied, reviewed by a physician of its choosing. (TR 15-19).

conglomerate fibrotic masses produced by the progressive massive fibrosis of complicated pneumoconiosis. Again, he saw the partially cavitated mass in the left lower zone.

Dr. Alexander noted that Mr. Roland had sufficient occupational exposure to produce a coal dust burden sufficient to cause pneumoconiosis. He noted that chest x-rays as early as 1973 found pneumoconiosis, with Category A pneumoconiosis first noted in 1980. Dr. Alexander found category C complicated pneumoconiosis on the x-rays he reviewed from 1999 to 2001. Additionally, the CT scans documented the multiple bilateral large masses with radiographic features characteristic of complicated pneumoconiosis. He noted that as progressive massive fibrosis occurs in pneumoconiosis, the actual profusion of small opacities decreases as they are incorporated into the formation of large opacities.

Dr. Alexander's final diagnosis was complicated pneumoconiosis, category C, 2/2, q, r; right pleural effusion; and right chest wall pleural thickening versus loculated fluid.

Dr. Alexander also prepared a report dated July 2, 2005, after reviewing an additional x-ray, as well as medical records (CX 8). It was clear to him, as well as to several other B readers, that Mr. Roland had simple pneumoconiosis of high profusion on his January 30, 1973 x-ray. He stated that being able to review x-rays from 1973, 1982, 1999, 2000, and 2001 allowed him to perform a serial assessment, which firmly established the diagnosis of complicated pneumoconiosis. According to Dr. Alexander, looking at the 1973 and 1982 x-rays side by side, the typical pattern and progression from simple to complicated pneumoconiosis can be clearly seen.

Dr. Alexander noted that on x-rays taken from 1973 to 1999, numerous interpreters, himself included, found q or r rounded opacities, of moderate to high profusion. But these small opacities were less apparent on later films, because when massive pulmonary fibrosis occurs in coalworkers' pneumoconiosis, the actual profusion of small opacities decreases as they are incorporated into the formation of large opacities. He cited to statements by Dr. Cole, in the NIOSH Study Syllabus for Classification of Radiographs of Pneumoconioses, that:

> A large opacity of pneumoconiosis is almost invariably associated with an unequivocal background of small pneumoconiotic opacities with a profusion level of category 1 or greater. As large opacities increase in size, however, they appear to incorporate surrounding small opacities. Moreover, as they enlarge, emphysematous changes in the surrounding lung become increasingly apparent. As a result, the profusion of small opacities surrounding a large opacity may appear to diminish over time and rarely may disappear entirely.

Again, Dr. Alexander explained that the large opacities in Mr. Roland's lungs developed over many years, starting as Category A at least by 1980, and progressing to Category C by 1999. Dr. Alexander again cited to Dr. Cole in the NIOSH study syllabus:

> The large opacities of pneumoconiosis change very slowly – usually over the course of years. Opacities representing tuberculosis, rheumatoid nodules and carcinomas change more rapidly – usually over the course of months. Moreover, the large opacities of pneumoconiosis appear to migrate toward the hilum as emphysematous changes progress. This is not seen in the case of other large opacities.

Dr. Alexander noted that in Mr. Roland's case, his chest x-rays, dating back to 1973, show simple pneumoconiosis with a background of small round opacities of high profusion. But the pattern and location of the large opacities shown on later x-rays are clearly much more consistent with the development of PMF, rather than tuberculosis, cancer, or granulomatous disease.

Based on the above, and his personal review of seven x-rays and two CT scans, Dr. Alexander disagreed with the readings by Dr. Wheeler, Dr. Scott, and Dr. Scatarige, and felt that they were incorrect in attributing the large pulmonary opacities to disease processes other than complicated pneumoconiosis.

Dr. Alexander prepared a report dated July 2, 2005, interpreting a December 9, 1982 x-ray, and excerpts from Dr. Wheeler's deposition (CX 8). Again, he stated that it was clear to him, as well as several other B readers, that Mr. Roland had simple pneumoconiosis of high profusion on the January 30, 1973 x-ray. He felt that his ability to review serial x-rays from 1973, 1982, 1999, 2000, and 2001 allowed him to make a serial assessment that firmly established the diagnosis of complicated pneumoconiosis. According to Dr. Alexander, in looking at the 1973 and 1982 x-rays side by side, the typical pattern and progression from simple to complicated pneumoconiosis is clearly seen.

Dr. Alexander noted that on x-rays from 1973 to 1999, ten physicians, the West Virginia Occupational Pneumoconiosis Board, and he all found q or r rounded opacities of moderate to high profusion. Again reviewing the January 30, 1973 x-ray, he stated that it showed small round opacities predominantly of the q size, with a profusion of 3/2 to 3/3. But these small opacities were less apparent on later films, because when PMF occurs in coalworkers' pneumoconiosis, the actual profusion of small opacities decreases as they are incorporated into the formation of large opacities.

Dr. Alexander repeated the statements he made in his earlier report, citing from Dr. Cole, and the NIOSH study syllabus, and disagreeing with the x-ray readings by Dr. Wheeler, Dr. Scott, and Dr. Scatarige.

Employer's X-ray Submissions

The Employer submitted rebuttal readings of each of the x-rays performed while Mr. Roland was hospitalized. In addition, the Employer submitted rebuttal readings of

each of the x-rays reviewed by Dr. Alexander. Finally, the Employer submitted its two affirmative x-ray readings. These readings are summarized below.

Dr. Wheeler and Dr. Scott, who are both dually qualified, read Mr. Roland's ***March 25, 1999 x-ray*** (EX 4, 11). Dr. Wheeler found this x-ray to be negative for pneumoconiosis. He noted an 8 cm. mass in the right mid and upper lung, a 10 X 3 cm. mass in the left mid and upper lung, and a 3.5 cm. mass in the left mid lung, compatible with conglomerate granulomatous disease, tuberculosis, or histoplasmosis. He also noted a few small nodules in the lateral right upper lung, and possibly a few in the mid lungs, compatible with granulomata, and probable minimal fibrosis in the left lower lateral lung.

Dr. Scott rated this x-ray as 0/1, r, q. He noted an 8 X 5.5 cm. mass in the right mid lung, a 10 X 3 cm. mass in the mid lung, and scars that extended from the masses to thickened pleura. Dr. Scott noted a few small granulomata in the right upper lung. He indicated that the changes were probably due to tuberculosis or unknown activity.

Dr. Scatarige, who is dually qualified, interpreted Mr. Roland's ***March 20, 2000 x-ray*** (EX 5). Dr. Scatarige found this x-ray to be negative for pneumoconiosis. He noted an 8 cm. mass in the right mid lung, a 4 cm. mass in the left mid lung, and a 3 cm. mass in the left upper lung, as well as a possible 2 cm. mass in the left lower lung. His diagnosis was metastasis, Wegener's, or doubtful tuberculosis. But he saw no small nodules of pneumoconiosis.

Dr. Scatarige and Dr. Scott reviewed Mr. Roland's ***December 20, 2000 x-ray*** (EX 6, 12). Dr. Scott found this x-ray to be negative for pneumoconiosis, with an 8 cm. mass in the right mid lung with associated thickened chest wall pleura, a 12 X 4 cm. mass in the left mid lung, and a small right pleural effusion, that represented probable granulomatous disease. He noted the lack of small rounded opacities to suggest silicosis or coal workers' pneumoconiosis.

Dr. Scatarige found this x-ray to be negative for pneumoconiosis, but he noted a 7 X 8 cm. mass in the right mid lung, masses in the left upper and mid lung, and a 2 cm. mass in the left lower lung. He "favored" metastasis, asking if there was a known primary cancer, or Wegener's. He stated that multiple primary cancers or tuberculosis were less likely, and that there were no small round opacities to suggest pneumoconiosis.

Dr. Scott read Mr. Roland's ***December 23, 2000 x-ray*** (EX 13). Dr. Scott found this x-ray to be negative for pneumoconiosis, but he noted bilateral mid lung masses that were probably granulomatous, posterior blunting due to effusion or fibrosis, and hyperinflation due to emphysema.

Dr. Scott also reviewed Mr. Roland's ***December 25, 2000 x-ray*** (EX 14). He stated that the digital format and small images were not authorized for an ILO reading. However, he noted that there were bilateral large mid lung masses with scarring extending to the pleura, with associated pleural thickening, most likely due to tuberculosis or unknown activity.

Dr. Wheeler and Dr. Scott reviewed Mr. Roland's ***May 24, 2001 x-ray*** (EX 9, 16). Dr. Wheeler found the x-ray to be negative for pneumoconiosis, but he noted a 7 cm. mass in the lower central right upper lung, a 4 cm. mass in the right and mid upper lung, a curved mass in the left mid and upper lung 11 cm. long and up to 3.5 cm. thick, and a 3 cm. mass in the left mid lung, all compatible with conglomerate granulomatous disease, tuberculosis, or histoplasmosis. He also noted smooth right lateral pleural fibrosis up to 1.5 cm. thick near the lower right scapula, and minimal bilateral lower lateral pleural fibrosis, as well as a few tiny linear scars, compatible with healed inflammatory disease. He stated that there were no obvious symmetrical background nodules on the CT scan to suggest that the masses were large opacities of coal workers pneumoconiosis.

Dr. Scott found this x-ray negative for pneumoconiosis, but noted a 10 cm. mass in the right mid lung, and a 12 X 4 cm. mass in the left mid lung, with linear scars extending to the pleura, and thickened pleura on the right. He stated that the changes were probably due to tuberculosis or unknown activity. But there was no background of small rounded opacities to suggest pneumoconiosis.

Dr. Scott and Dr. Scatarige interpreted Mr. Roland's ***June 1, 2001 x-ray*** (EX 10, 17). Dr. Scatarige found no evidence of pneumoconiosis, but he noted multiple bilateral lung masses that could be metastases. He also noted right pleural effusion, and small left pleural effusion, but no small round opacities to suggest pneumoconiosis.

Dr. Scott found this x-ray negative for pneumoconiosis, but he noted a 9 X 5.5 cm. mass in the right mid lung with associated lateral thickened pleura, as well as an 11 X 3 cm. left mid to upper lung mass, and a 3 cm. mass in the left lower lung. He stated that these changes were probably granulomatous disease, and possibly tuberculosis. But there was no background of small rounded opacities to suggest pneumoconiosis.

The Employer also submitted interpretations of CT scans performed while Mr. Roland was hospitalized. Dr. Wheeler reviewed Mr. Roland's ***December 23, 2000 CT scan*** (EX 7). He noted 6 and 4 cm. masses in the right mid and upper lung involving the hilum, with calcifications and areas of low density, compatible with conglomerate granulomatous disease and necrosis. He also noted a 3 cm. diameter elongated mass in the left mid and upper lung, containing small calcified granulomata, and areas of low density, compatible with conglomerate tuberculosis of histoplasmosis, with probable central necrosis.

Dr. Wheeler also found a 4 cm. mass in the left lower lung, with a 1.5 cm. partly thin walled cavity, compatible with inflammatory disease or cavitary cancer. He noted 1 cm. nodules in the lateral right upper lung, compatible with granuloma more likely than tumor, and moderate emphysema seen best in the right lung. Dr. Wheeler also found areas of bilateral pleural fibrosis, and scattered peripheral linear scars from healed inflammatory disease. He felt that an exact diagnosis needed to be made to assure proper therapy.

Dr. Wheeler reviewed the ***February 26, 2001 CT scan*** films (EX 15). He noted a well defined mass in the right upper and mid lung, involving the right hilum, and a curved mass in the left mid and upper lung near the hilum, with areas of low density suggesting necrosis, and a "probable" few tiny calcified granulomata, especially in the right upper lung mass, compatible with conglomerate tuberculosis or histoplasmosis. Dr. Wheeler also described a 4 cm. mass partly in a thin walled cavity, compatible with inflammatory disease or cancer.

Dr. Wheeler noted a 2.5 cm. mass in the left cardiophrenic angle, a 2 cm. mass in the left lateral CPA, a 1 cm. mass in the lateral subapical right upper lung, and a 1 cm. mass in the posterior right mid lung and posterior inferior left lower lung, which he felt were compatible with granulomata more likely than metastases.

According to Dr. Wheeler, there were no small background nodular infiltrates to indicate pneumoconiosis, and only very rarely were all of the small nodules merged into large opacities. He felt that an exact diagnosis was needed, which would require a transbronchial biopsy of the right upper lung mass that involved the hilum.

Dr. Wheeler added a postscript, stating that large opacities of this size typically appeared in drillers who worked unprotected during and before World War II.

Dr. Scatarige also reviewed Mr. Roland's ***February 26, 2001 CT scan*** (EX 8). He found no small round opacities to suggest pneumoconiosis. However, he noted multiple bilateral necrotic lung masses, largest in the right upper lung at 7 cm. and a 3 cm. cavitary mass in the left lower lung. His diagnosis was necrotic metastases, granulomatous disease, or Wegener's.

Dr. Wheeler's Deposition Testimony

As discussed at the hearing, and in a post-hearing teleconference, the Employer was provided the opportunity to have Dr. Alexander's report and x-ray interpretations reviewed. The Employer submitted no such written report, but instead on May 18, 2005 took Dr. Wheeler's deposition, where he discussed his interpretation of the x-rays and CT scans that were also reviewed by Dr. Alexander (EX 18). But in his relatively brief testimony, Dr. Wheeler made no reference to Dr. Alexander's report, and there is no indication that Dr. Wheeler ever reviewed it. Rather, Dr. Wheeler's testimony is essentially a rehash of his own interpretations of the x-rays.

In response to a question about how he was able to distinguish masses from large opacities, Dr. Wheeler stated that large opacities typically should be coalescent small opacities, which are usually rounded nodules of one size or another. They should not have calcifications, which are a strong indication of granulomatous disease. He stated that what indicates the presence of large opacities is a background nodularity of relatively high profusion, usually q and r, because it is easier to make a large opacity out of bigger

nodules than out of tiny nodules. Here, there were none of the larger small opacities, and no symmetrical nodular patterns as a background for the masses.

Dr. Wheeler stated that the masses in this case also had calcification, which strongly favors granulomatous disease. In addition, there were areas of necrosis; on one of the x-rays, he was not sure if was a cavitary cancer, or a cavitary inflammatory process. In discussing the February 26, 2001 CT scan, Dr. Wheeler stated that the differential diagnosis for the mass presumably was inflammatory, but he could not exclude metastases. He felt that an exact diagnosis was needed.

According to Dr. Wheeler, the typical granulomatous disease to consider is tuberculosis and histoplasmosis; another is sarcoid, which tends to be more common in Blacks in America. Dr. Wheeler stated:

> So, assuming he was not a driller working unprotected during World War II or before World War II, it's unlikely that these are large opacities, because my experience has been the group of people who have the large opacities were drillers working unprotected in high dust exposures and they developed large opacities. That was during and before World War II.

EX 18 at 14.

Dr. Wheeler stated that his most likely diagnosis in this case was some form of granulomatous disease. He stated: "I can tell you that the sun doesn't set on mass lesions in my hospital many times before an exact diagnosis is made."[4]

According to Dr. Wheeler, histoplasmosis is more common than tuberculosis, but tuberculosis more commonly involves the upper lungs.

Dr. Wheeler discussed the fact that some of the x-rays had small opacities that were not rounded. But he did not see small round nodules on any of the films as the "primary" small opacity, and in this case he assumed that it was some disease process other than silicosis or coal workers' pneumoconiosis. Dr. Wheeler stated that what he wanted to see were background nodules in the mid and upper lung zones, generally in the central portions, which do not contain calcium, and do not have cavities.

Medical Reports

Dr. C.P. Vasudevan

Dr. C.P. Vasudevan was Mr. Roland's treating physician; he attended Mr. Roland during his hospitalizations at the Bluefield Regional Medical Center. He submitted a letter dated December 21, 2004 (CX 4, DX 20), stating that Mr. Roland died of chronic

[4] Dr. Wheeler commented several times on the lack of an exact diagnosis of these masses. Apparently, he was unaware that Mr. Roland was repeatedly diagnosed by his treating physicians as suffering from severe complicated pneumoconiosis.

respiratory failure and acute exacerbation of chronic obstructive pulmonary disease, caused by his complicated coal workers' pneumoconiosis/progressive massive fibrosis. According to Dr. Vasudevan, Mr. Roland's primary condition of complicated coal workers' pneumoconiosis was due to his thirty year history of work in the coal mines, and was progressive, as evidenced by the progressive impairment of lung function and hypoxemia. It led to several hospitalizations with acute respiratory distress and failure, and finally his death.

The record includes treatment notes from Dr. Vasudevan, in which are included the results of a pulmonary function test performed while Mr. Roland was at the Bluefield Hospital on March 2, 2001. The FVC was 1.94/2.08; the FEV1 was 1.06/1.12; and the MVV was 23/24. Mr. Roland's height was reported as 67 inches; he was 75 years old. Dr. Vasudevan had Mr. Roland undergo pulmonary function testing on November 1, 1999, which produced an FVC of 2.3/2.53; and an FEV1 of 1.21/1.31. There are also arterial blood gas test results from an admission at the Bluefield Regional Medical Center on September 2, 2000.

Bluefield Regional Medical Center

Mr. Roland was admitted to the Bluefield Regional Medical Center on January 3, 2002, and passed away on January 9, 2002 (DX 18). He was noted to have COPD and progressive pulmonary fibrosis; he was admitted with a headache and worsening shortness of breath. Dr. Dennis Kolokolo noted that Mr. Roland's respiratory distress had increased remarkably, and he was quite short of breath on minimal exertion; he needed assistance to get out of bed. Repeated CT scans of his chest showed a large right sided pleural effusion with moderate layering. Mr. Roland was supposed to be transferred to the University of Virginia for further treatment, but he passed away at the Bluefield Regional Medical Center.

Mr. Roland was admitted to the Bluefield Regional Medical Center on February 24, 2001 for complaints of increasing shortness of breath, chills with fever, and productive couth, and discharged on March 2, 2001 (DX 15). A CT angiogram of the chest showed conglomerate masses of complicated coal workers' pneumoconiosis, and a pleural effusion that had been partially drained at Charlottesville. Mr. Roland was put on oxygen. His discharge diagnosis was acute respiratory failure; acute exacerbation of chronic obstructive disease complicated by coal workers' pneumoconiosis; and history of pulmonary embolism. Pulmonary function testing showed moderately reduced forced vital capacity, and severely reduced forced expiratory volume.

Mr. Roland was admitted to the Bluefield Regional Medical Center on December 29, 2001, and discharged on December 31, 2001 (DX 17). Dr. Brinegar's discharge diagnosis was exacerbation of chronic obstructive pulmonary disease, and coal workers' pneumoconiosis.

Mr. Roland was transferred from Bluefield Regional Medical Center to University of Virginia on December 21, 2000 (DX 14). His discharge diagnosis was acute

respiratory distress and failure; acute exacerbation of chronic obstructive airway disease; complicated coalworkers' pneumoconiosis with progressive massive fibrosis; and possible unstable angina. He had come to the emergency room with progressive shortening of breath, and pain in his upper abdomen with exertion. He had been hospitalized there before for acute respiratory failure, precipitated by acute exacerbation of chronic obstructive airway disease. Dr. Vasudevan noted that he had been on oxygen and breathing treatments for some time.

Mr. Roland was admitted to the Bluefield Regional Medical Center on September 1, 2000 with severe shortness of breath and tightness in the chest, and discharged the following day (DX 13). His discharge diagnosis was acute respiratory distress, and acute exacerbation of chronic obstructive pulmonary disease and complicated pneumoconiosis.

Mr. Roland was admitted to the Bluefield Regional Medical Center on March 17, 2000 with worsening shortness of breath, and discharged on March 20, 2000 (DX 12). His discharge was acute respiratory failure, acute exacerbation of chronic obstructive airway disease, and complicated coalworkers' pneumoconiosis.

Mr. Roland was admitted to the Bluefield Regional Medical Center on October 6, 1999 with worsening shortness of breath, and discharged on October 12, 1999 (DX 11). His discharge diagnosis was acute respiratory distress; acute exacerbation of chronic obstructive airway disease secondary to purulent bronchitis; and complicated coalworkers' pneumoconiosis. The x-ray done in the emergency room showed bilateral conglomerate masses, with background changes of pneumoconiosis, consistent with complicated coalworkers' pneumoconiosis.

Dr. D.L. Rasmussen

Dr. D.L. Rasmussen reviewed medical records from Mr. Roland's hospitalizations in the last years of his life, as well as the death certificate (CX 3). He indicated that Mr. Roland died of respiratory failure on January 9, 2002; he was previously hospitalized and treated for respiratory failure on many occasions between 1999 and the date of his death. On every occasion, his respiratory failure was considered to be related to his underlying complicated coalworkers' pneumoconiosis. Dr. Rasmussen noted that during one hospitalization, Mr. Roland was found to have evidence of pulmonary infarction in the left lower lung zone, but he was not felt to have embolization during other episodes of respiratory failure. Mr. Roland was evaluated for cardiac disease, but had no evidence of left ventricle disease, although it was felt that he had one mild area of ischemia on one isotope study. He had pleural effusion that was attributed to right heart failure secondary to chronic severe lung disease.

According to Dr. Rasmussen, the basis for Mr. Roland's diagnosis of complicated coalworkers' pneumoconiosis was his significant history of coal mine dust exposure, and his progressive x-ray abnormalities, beginning in 1973. Dr. Rasmussen reviewed these findings, also noting that CT scans were consistently interpreted as showing conglomerate masses. Mr. Roland's pulmonary function studies showed progressive

ventilatory impairment, from mild to moderately severe. He developed periodic respiratory failure with hypoxia.

Dr. Rasmussen pointed out that Mr. Roland's obstructive lung disease could not be attributed to smoking, as he had a history of very limited smoking, mostly pipe or occasional cigar smoking. Nor was there ever a suspicion of pulmonary tuberculosis. Dr. Rasmussen acknowledged that while neither tuberculosis nor cancer could be one hundred percent excluded based on the x-ray findings, the fact that Mr. Roland had classical radiographic features, with progressively severe impairment in lung function, virtually excluded any other cause for his disabling lung disease and death besides coal workers' pneumoconiosis.

In Dr. Rasmussen's opinion, Mr. Roland suffered from complicated coalworkers' pneumoconiosis, or progressive massive fibrosis, as a consequence of his 31 years of coal mine employment, much of it at the face and before the institution of dust suppression measures in the coal mines, and his death was due primarily to his complicated coalworkers' pneumoconiosis.

Dr. Rasmussen submitted a report dated June 20, 2005, after reviewing additional medical records (CX 7). He indicated that these records did not change his opinion, that Mr. Roland suffered from progressively severe, and ultimately fatal, coalworkers' pneumoconiosis. Dr. Rasmussen indicated that he was not able to judge why there was a difference in interpretations as between the radiologists from the area, and those from Johns Hopkins University. He acknowledged that Dr. Wheeler, Dr. Scott, and Dr. Scatarige were very highly qualified and experienced radiologists, who had been involved in reading films for pneumoconiosis for many years. Dr. Rasmussen noted that these physicians believe that only a small percentage of films they view show evidence of pneumoconiosis, in contrast with a higher percentage of positive interpretations by B readers from NIOSH. But he was aware of no reports of correlation of radiographic and pathologic findings among subjects whose films had been interpreted by either group. He did state that he was aware of a number of cases in which the Baltimore physicians interpreted x-rays as negative, in contrast with local B readers, where definite pneumoconiosis, including complicated pneumoconiosis, was subsequently found on autopsy. He stated that there was no basis for assuming that radiologists from Johns Hopkins more accurately interpret x-rays for pneumoconiosis.

Dr. Rasmussen also noted that x-rays, and even CT scans, are not infallible. Citing to published studies, he stated that it was well known that significant pneumoconiosis, even severe grades of small macular or micronodules, may be found without radiographic changes. According to Dr. Rasmussen, a diagnosis of complicated pneumoconiosis is made on the basis of a large opacity, in the presence of simple pneumoconiosis. But it is known that complicated pneumoconiosis occurs without diffuse radiographic abnormalities. He stated that small round opacities of simple pneumoconiosis also become less and less visible as emphysema progresses. Thus, miners whose x-rays initially showed rounded densities may have a progression of irregular opacities, which eventually become more apparent and replace rounded

opacities. As such, complicated pneumoconiosis can occur on a background of irregular, not rounded opacities.

According to Dr. Rasmussen, there have been no epidemic findings of tuberculosis in the rural Appalachian coal fields, as there apparently was in Baltimore or other large cities. Nor have autopsies related more than a very small number of aggressive histoplasmosis.

Again, Dr. Rasmussen stated that Mr. Roland had numerous x-rays interpreted by qualified B readers that showed the presence of a high profusion of rounded opacities, beginning as early as 1973, with the subsequent appearance and progressive increase in the size of large opacities, quite characteristic of complicated pneumoconiosis. He saw no medical reason to doubt the diagnosis of complicated pneumoconiosis. He noted that this conclusion was buttressed by the opinion of Mr. Roland's treating pulmonologist, Dr. Vasudevan, who believed that complicated pneumoconiosis was the ultimate cause of Mr. Roland's death.

Dr. Rasmussen discussed Dr. Crisalli's conclusion that Mr. Roland's death was due to some undiagnosed intracranial disease. He noted that Dr. Crisalli questioned the diagnosis of complicated pneumoconiosis, pointing to the fact that complicated pneumoconiosis does not cause pleural effusion, and questioning Dr. Rasmussen's statement that the pleural effusion was apparently attributed to right heart failure secondary to chronic severe lung disease. Dr. Rasmussen cited to the December 30, 2000 discharge summary, agreeing that complicated pneumoconiosis did not cause the pleural effusion, which was due to severe destruction of lung tissue, with resultant right ventricular failure.

With respect to Dr. Crisalli's opinion that the condition that caused the headache might have been responsible for Mr. Roland's death, Dr. Rasmussen noted that there are actually multiple possible immediate causes of death, but many of them, including central nervous system diseases, represent a combination of respiratory failure with some superimposed event. According to Dr. Rasmussen, whatever caused Mr. Roland's headache cannot be determined. But there was ample evidence of a progressively severe lung disease that had the capability of causing death at any moment, especially during Mr. Roland's last hospitalization.

Again, Dr. Rasmussen concluded that Mr. Roland died as a result of classically progressive lung tissue destruction as a consequence of complicated pneumoconiosis that arose from his coal mine employment.

University of Virginia Health Sciences Center

The exhibits include records from the University of Virginia Health Sciences Center, where Mr. Roland was treated in December 2000 (CX 2). Mr. Roland was admitted on December 22, on transfer from Bluefield Hospital, for workup of a possible

cardiac etiology. At that time, Mr. Roland had a one year history of worsening dyspnea on exertion, and a three year history of decreased activity due to dyspnea. He had been on home oxygen for six months. The section for Mr. Roland's past history noted that he had COPD secondary to pneumoconiosis and progressive massive fibrosis; no history of tuberculosis exposure; and a left breast mass with a negative mammogram.

Computerized tomography studies showed evidence of multiple small pulmonary emboli in the left lower lobe, bilateral patchy alveolar and interstitial infiltrates, moderate right pleural effusion, trivial left sided effusion, and small pericardial effusion; there were upper lobe masses consistent with massive pulmonary fibrosis, scattered nodules, some with fasciculated borders, and loculated mid thorax anterior effusion. Although chest films were requested, they did not arrive by the date of discharge, for comparison to further elucidate the lung process. But it was thought most likely to be massive pulmonary fibrosis secondary to the progression of his pneumoconiosis.

The principal diagnosis was left lower lobe pulmonary embolus; massive progressive fibrosis on pneumoconiosis; right heart failure; chronic obstructive pulmonary disease exacerbation; and right exudative pleural effusion, negative for malignancy.

Chest x-rays performed on December 23, 2000 showed focal masses in both lungs, right greater than left. They were thought to represent pulmonary infiltrates from pneumonia, metastatic or primary carcinoma, or a sequela of Mr. Roland's hemoptysis. There was a baseline reticular pattern throughout the lungs. It was felt that the findings could represent progressive massive fibrosis in a person with pneumoconiosis. X-rays performed later that day showed no interval change. A third set of x-rays performed that evening again showed bilateral lung masses with a broad differential, and no significant change, except for perhaps slightly increased opacity in the left upper lung.

Mr. Roland underwent a CT scan on December 23, 2000. Multiple, predominantly round masses were identified within the perihilar regions and upper lobes. The largest mass measured approximately 5.1 by 5.1 cm. in the right upper lobe. Many of the masses had low density centrally, with punctuate calcification within the largest mass in the right upper lobe. Some of the masses had pleural tethering. Interstitial density was identified throughout most of the remainder of the lungs, with sparing of the superior segments of the lower lobes and the right lung base. There was a suggestion of honeycombing at the posterior periphery of the left lung base. Additionally, multiple poorly defined 1-2 cm. nodules were identified bilaterally. There was questionable cavitation in a left lower lobe nodule. There was a moderate sized right pleural effusion, and a small amount of pleural thickening or fluid in the right anterolaterally.

Dr. Hazard's impression was multiple predominantly round masses in the perihilar regions and upper lung zones, with punctuate calcification in one of the masses, and a suggestion of cavitation in another mass. He felt the findings were suspicious for metastatic disease. He stated that progressive massive fibrosis would also be in the differential diagnosis, although the imaging features were not classic. According to Dr.

Hazard, the reticulonodular densities throughout most of the lung were consistent with Mr. Roland's history of pneumoconiosis.

Mr. Roland underwent a CT scan of his thorax on December 23, 2000. Dr. Sabah D. Butty reviewed the study, noting a heart of normal size, with small pericardial effusion. There were bilateral pleural effusions, much greater on the right. He identified a loculated pleural effusion in the mid right hemithorax. Dr. Butty noted several bilateral varying sized masses, some spiculated, and a single large central mass in each hemithorax. The largest mass, centered about the right hilum, had patchy, inhomogeneous enhancement, and measured approximately 6.2 cm. in greatest diameter. There were several pulmonary emboli in arterial branches in the left lower pulmonary lobe. Dr. Butty stated that without previous imaging for comparison or correlation, it was uncertain whether the masses were of neoplastic origin, or represented progression of Mr. Roland's known pneumoconiosis.

An x-ray performed the morning of December 23, 2000, and reviewed by Dr. Drew Lambert, showed mixed reticulonodular and confluent opacities in the bilateral lung fields, consistent with Mr. Roland's known chronic lung disease. There was interval improvement in bilateral aeration, and no new focal opacities.

Prudich Medical Center

The exhibit file includes treatment records from Prudich Medical Center from March 1999 to (CX 1). On March 25, 1999, Dr. Sheth noted Mr. Roland's four month history of cough, congestion, and exertional dyspnea; he was worried about his history of worsening exertional dyspnea over the past year. He felt that, in connection with his cough and history of mild pneumoconiosis, his pneumoconiosis could be getting worse. He also needed to rule out pneumonia or CHF, although his clinical examination did not suggest these. An x-ray performed that same day was interpreted by Dr. Stephen P. Raskin, who found complicated coalworkers' pneumoconiosis, with progressive massive fibrosis in the upper zones, and interstitial lung disease. There was no consolidation or effusion.

At a follow-up visit on March 29, 1999, Dr. Sheth discussed with Mr. Roland the results of his x-ray, which confirmed coalworkers' pneumoconiosis with progressive massive fibrosis in the upper zones. Dr. Sheth felt that the x-ray picture of worsening pneumoconiosis was consistent with Mr. Roland's history of significant deterioration in exercise tolerance for the last year or two. Although Dr. Sheth noted that there was no treatment, he advised Mr. Roland to see a pulmonary specialist.

Dr. Robert J. Crisalli

Dr. Crisalli reviewed medical records at the request of the Employer, and prepared a report dated May 19, 2005 (EX 19). He concluded that there was no question that Mr. Roland had severe lung disease. But he felt that the lesions as described by radiologists at Johns Hopkins and the University of Virginia were suggestive of non-

occupational related densities. He indicated that "possible diagnoses" included metastatic lung disease, and granulomatous lung disease. He noted that the radiologists indicated that a biopsy was necessary to make a diagnosis, which indicated to Dr. Crisalli that the radiographic appearance of the lesions was not typical of occupational pneumoconiosis, and that the radiologists were describing lesions that could be cancer, or granulomatous disease.

Dr. Crisalli acknowledged that he did not know the circumstances surrounding Mr. Roland's death. He stated that Mr. Roland's primary problem in his final hospital admission was headache, and it was "very possible" that his death was related to an intracerebral event, but not in any way related to his lung disease.

Dr. Crisalli placed most reliance on the x-ray interpretations by the physicians from Johns Hopkins Hospital and the University of Virginia, who found that none of the changes suggested pneumoconiosis, but were more consistent with granulomatous lung disease or metastatic lung disease. He also noted that the physicians from the University of Virginia commented that the lesions were spiculated, suggesting malignancy, and that their appearance was not typical for pneumoconiosis.

According to Dr. Crisalli, there was nothing in the records to indicate that Mr. Roland died due to his pulmonary disease, although he did have significant pulmonary disease. He noted that in the last discharge summary from Bluefield Hospital, the physician stated that Mr. Roland's respiratory distress had increased remarkably, and that he had a large right-sided pleural effusion that could not be drained. Dr. Crisalli stated that it was "likely" that Mr. Roland's increased shortness of breath was related to this pleural effusion; pneumoconiosis does not cause pleural effusions. Dr. Crisalli was also concerned about the indication in the death summary, that headache was a significant condition contributing to Mr. Roland's death. Dr. Crisalli felt that Mr. Roland's death was "more related to whatever was causing his headache and not related to his chronic respiratory disease."

DISCUSSION

A. Existence of Pneumoconiosis

Section 718.202 provides four means by which pneumoconiosis may be established. Under § 718.202(a)(1), a finding of pneumoconiosis may be made on the basis of the x-ray evidence. In this case, there are numerous narrative reports of x-rays performed while Mr. Roland was hospitalized between 1999 and his death in 2001. Almost without exception, the physicians who reviewed these x-rays concluded that Mr. Roland had severe and advanced complicated pneumoconiosis, with large conglomerate masses in both lungs.

Dr. Alexander, who is a dually qualified radiologist, reviewed a series of x-rays and CT scans covering this period, between 1999 and 2001, noting the presence of large, bilateral masses in Mr. Roland's lungs, on a background of small opacities that decreased

in profusion as they were incorporated into the formation of large opacities. In his second report, Dr. Alexander also reviewed x-rays dating back to 1973, which he felt firmly established the diagnosis of complicated pneumoconiosis. He noted that the earlier x-rays had been interpreted by many readers, himself included, to show rounded opacities of moderate to high profusion. However, these small opacities were not as apparent on later films, as they became incorporated into the formation of large opacities. Looking at the longitudinal series of x-rays, Dr. Alexander felt that they clearly showed the typical pattern and progression from simple to complicated pneumoconiosis.

On the other hand, Dr. Scott, Dr. Scatarige, and Dr. Wheeler, who are also dually qualified, uniformly interpreted the x-rays performed between 1999 and 2001 as negative for pneumoconiosis. If I were to rely strictly on the numerical superiority of the negative ILO readings by well qualified readers, I would conclude that the x-ray evidence is not sufficient to support a finding of pneumoconiosis. Thus, Dr. Groten, who administered the October 6, 1999 x-ray, did not make a specific finding of pneumoconiosis, but he noted density in the right and left lungs, as well as the possibility of bilateral pulmonary masses.

Four x-rays administered over the year 2000 were reviewed by Dr. Rao, Dr. Shahan, and Dr. Miller, who consistently noted conglomerate densities in both lungs, and diagnosed severe complicated pneumoconiosis, or in Dr. Shahan's case, chronic severe interstitial disease. Similarly, with respect to the three x-rays that Mr. Roland underwent in 2001, Dr. Cappiello, Dr. Fowler, and Dr. Miller all noted the presence of large bilateral masses, and diagnosed severe complicated pneumoconiosis. Dr. Aycoth, who reviewed Mr. Roland's January 6, 2002 x-ray, noted progressive massive fibrosis and conglomerate lung masses of complicated pneumoconiosis.

Dr. Cappiello also reviewed the CT scan that Mr. Roland had on February 26, 2001, noting multiple conglomerate masses of complicated pneumoconiosis. Dr. Cappiello also noted the presence of a cavity that had undergone necrosis and spontaneously drained, which he also saw on Mr. Roland's May 24, 2001 x-ray.

Thus, with the exception of the interpretations by Dr. Scott, Dr. Scatarige, and Dr. Wheeler, the physicians who reviewed Mr. Roland's x-rays uniformly made findings consistent with pneumoconiosis.

I rely on the detailed, well-supported, and persuasive review of the x-ray and CT scan evidence by Dr. Alexander, as supported by the narrative interpretations discussed above, and find that they outweigh the negative interpretations by Dr. Scott, Dr. Scatarige, and Dr. Wheeler. Thus, I find that the Claimant has established that Mr. Roland had pneumoconiosis by a preponderance of the persuasive x-ray evidence.

Under § 718.202(a)(2), a finding of pneumoconiosis may be made on the basis of biopsy or autopsy evidence. There is no such evidence in this case, and thus, the Claimant has not established pneumoconiosis under § 718.202(a)(2).

The Claimant can also establish the existence of pneumoconiosis with reasoned medical opinions. Under § 718.202(a)(4), a determination of the existence of pneumoconiosis may be made if a physician exercising reasoned medical judgment, notwithstanding a negative x-ray, finds that the miner suffers from pneumoconiosis as defined in §718.201.

The hospital and treatment records document a series of hospital admissions for respiratory distress, and repeated diagnoses of severe and progressive complicated pneumoconiosis. These diagnoses were supported by testing, such as the CT angiogram performed at the Bluefield Regional Medical Center in February 2001, which showed conglomerate masses of complicated pneumoconiosis, and the x-ray performed in the Bluefield emergency room in October 1999, which showed bilateral conglomerate masses, with background changes of pneumoconiosis, consistent with complicated coalworkers' pneumoconiosis.

Dr. Rasmussen, who reviewed Mr. Roland's medical records, noted his significant history of coal dust exposure, much of it at the face and before the institution of dust suppression measures, his progressive x-ray abnormalities, beginning in 1973, his findings on CT scans, which were consistently interpreted as showing conglomerate masses, his progressively worsening ventilatory impairment, and development of respiratory failure with hypoxia. He indicated that Mr. Roland had a very limited history of smoking, and no suspicion of tuberculosis. According to Dr. Rasmussen, Mr. Roland's classic radiographic features, and his progressively severe impairment in lung function, virtually excluded any cause for his disabling lung disease and death other than complicated coal workers' pneumoconiosis.

In his supplemental report, Dr. Rasmussen stated that complicated pneumoconiosis can occur without diffuse radiographic abnormalities, and that the small round opacities of simple pneumoconiosis become less and less visible as emphysema progresses. Thus, complicated pneumoconiosis can occur on a background of irregular, and not rounded, opacities. In Mr. Roland's case, numerous readers interpreted his x-rays as showing a high profusion of rounded opacities as early as 1973, with the subsequent appearance and progressive increase in size of large opacities, quite characteristic of complicated pneumoconiosis.

Dr. Vasudevan, Mr. Roland's treating physician, attended him when he was hospitalized at the Bluefield Regional Medical Center. He also submitted a letter stating that Mr. Roland died from chronic respiratory failure and acute exacerbation of chronic obstructive pulmonary disease, due to his complicated coal workers' pneumoconiosis. He indicated that this condition was due to Mr. Roland's thirty year history of work in the mines, and it was progressive, as evidenced by progressive impairment of lung function and hypoxemia. Dr. Vasudevan's treatment notes include results from pulmonary function and arterial blood gas testing documenting this decline.

In contrast, Dr. Crisalli, who reviewed medical records, conceded that Mr. Roland had severe lung disease, but that the lesions as described by the radiologists at Johns

Hopkins and the University of Virginia were suggestive of non-occupational related densities. The fact that these radiologists felt that a biopsy was necessary for diagnosis indicated to him that the appearance of the lesions was not typical of occupational pneumoconiosis, but could be cancer or granulomatous disease.

Dr. Crisalli did not explain why he put the most reliance on the x-ray interpretations by the physicians from Johns Hopkins and the University of Virginia. He seized on a notation from physicians at the University of Virginia that the lesions were spiculated, suggesting malignancy, to conclude that their appearance was not typical for pneumoconiosis. Mr. Roland underwent several x-ray and CT scans at the University of Virginia in December 2000. While there are notations of carcinoma as a differential diagnosis, the x-ray and CT findings overwhelmingly support a conclusion of complicated pneumoconiosis. Moreover, Dr. Crisalli's reliance on a conclusion that the lesions were spiculated is misleading. The only reference to spiculated masses is in Dr. Butty's review of the December 23, 2000 CT scan of Mr. Roland's thorax, where he noted several bilateral varying sized masses, **some** spiculated, and a single large central mass in each hemithorax. Dr. Butty did not have previous x-rays for comparison, and was uncertain if the masses were neoplastic, or represented a progression of Mr. Roland's known pneumoconiosis.

Thus, CT studies on December 22, 2000 showed upper lobe masses consistent with massive pulmonary fibrosis, secondary to the progression of Mr. Roland's pneumoconiosis. X-rays performed the next day, which showed focal masses in both lungs, were felt to represent progressive massive fibrosis in a person with pneumoconiosis. A CT scan the same day found multiple, predominantly round masses in both lobes, and multiple poorly defined 1-2 cm. nodules. Dr. Hazard, who reviewed the CT scan, felt that these findings were suspicious for metastatic disease, but that progressive massive fibrosis would also be in the differential diagnosis, although the imaging features were not classic. He also felt that the reticulonodular densities throughout most of Mr. Roland's lungs were consistent with his history of pneumoconiosis. An X-ray performed that same date showed mixed reticulonodular and confluent opacities, consistent with Mr. Roland's known chronic lung disease.

Finally, Dr. Alexander reviewed Mr. Roland's x-ray and CT scan films over a long period of time, and described persuasively and in great detail how they were led to a diagnosis of severe complicated pneumoconiosis. Dr. Alexander described the progression of Mr. Roland's lung masses over time, starting as simple pneumoconiosis with a high profusion of small rounded opacities, and progressing over time to numerous large opacities, consistent with the incorporation of the small rounded opacities into the formation of large opacities. Dr. Alexander relied on the NIOSH syllabus for classification of x-rays for pneumoconiosis, which was consistent with the progression of Mr. Roland's radiographic findings.

I also note that Dr. Alexander described Mr. Roland's February 26, 2001 CT scan as showing large masses with smooth **non-spiculated** well defined margins, central low attenuation areas, and internal calcifications, completely characteristic of the

conglomerate fibrosis found in complicated pneumoconiosis. There was also a suggestion of a central cavitation, which can occur in conglomerate masses of pneumoconiosis.

I place the greatest reliance on the reports by Dr. Alexander and Dr. Rasmussen, which are thorough and well-reasoned, and supported by the hospital treatment records, the report and records from Dr. Vasudevan, and the x-rays and CT scans performed while Mr. Roland was hospitalized. On the other hand, I accord no weight to the opinions by Dr. Crisalli, who did not review any of the radiological evidence or examine Mr. Roland, who provided no basis for his blind reliance on the readings by radiologists from Johns Hopkins and the University of Virginia, and whose selective discussion of the voluminous medical evidence did not discuss the progression of Mr. Roland's radiological findings over the years, or how those findings did or did not correlate with established criteria for a finding of pneumoconiosis, complicated or otherwise.

Nor am I swayed by the "medical report" of Dr. Wheeler, who essentially regurgitated his x-ray and CT scan interpretations. Dr. Wheeler did not address Dr. Alexander's report, or any of the voluminous medical evidence other than the x-rays and CT scans, nor did he cite to any studies or medical literature to support his conclusory statements. In contrast, Dr. Alexander's report was professional, well-reasoned and supported, and eminently persuasive

I find that the medical opinion evidence overwhelmingly establishes that Mr. Roland suffered from coalworkers' pneumoconiosis that had progressed at the time of his death to severe complicated pneumoconiosis. Therefore, the Claimant has established the existence of pneumoconiosis on the basis of the medical opinion evidence.

Finally, as required by the Fourth Circuit Court of Appeals, I have weighed all of the evidence on the issue of the existence of pneumoconiosis together, like and unlike, and I conclude that it establishes, again overwhelmingly, that Mr. Roland suffered from severe and progressive pneumoconiosis.[5]

Death Due to Pneumoconiosis

Even though Claimant has proven her husband had pneumoconiosis and the disease arose from his many years of coal mine employment, she must still establish that his death resulted from pneumoconiosis. I find that she has satisfied this burden.

Since the claim was filed after January 1, 1982, the issue of death due to pneumoconiosis is governed by § 718.205(c), as amended, which states, in pertinent part:

[5] Section 718.203(b) allows a rebuttable presumption of a causal connection where a miner worked in coal mines for more than 10 years and suffered from pneumoconiosis. No evidence has been submitted to overcome this presumption. Thus, I find Claimant has established a causal connection between her husband's pneumoconiosis and his coal mine employment.

> For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:
>
> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
>
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> (3) Where the presumption set forth at § 718.304 is applicable.
>
> (4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.
>
> (5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

20 C.F.R. § 718.205(c).

In *Eastern Associated Coal Corporation v. Director, OWCP,* 220 F.3d 250 (July 12, 2000), the Fourth Circuit discussed the three different ways set forth in the statute to establish the existence of statutory complicated pneumoconiosis in order to invoke the irrebuttable presumption at § 718.304, and noted that in applying the standards set forth in each prong,

> [O]ne must perform equivalency determinations to make certain that regardless of which diagnostic technique is used, the same underlying condition triggers the irrebuttable presumption.

Id. at 255, 256, *citing Double B Mining, Inc., v. Blankenship,* 177 F.3d 240, 243 (4th Cir. 1999). Additionally, the Court stated that

> "[B]ecause prong (A) sets out an entirely objective scientific standard" –i.e. an opacity on an x-ray greater than one centimeter –x-ray evidence provides the benchmark for determining what under prong (B) is a "massive lesion" and what under prong (C) is an equivalent diagnostic result reached by other means.

Id. at 256, citing Double B Mining at 243.

Although the Court acknowledged that a finding of statutory complicated pneumoconiosis may be based on evidence presented under a single prong, the Court also

noted that the ALJ must review the evidence under each prong for which relevant evidence is presented, to determine if complicated pneumoconiosis is present. The Court stated that:

> Evidence under one prong can diminish the probative force of evidence under another prong if the two forms of evidence conflict. Yet, "a single piece of relevant evidence," *Lester [Lester v. Director, OWCP], 993 F.2d at 1145,* can support an ALJ's finding that the irrebuttable presumption was successfully invoked if that piece of evidence outweighs conflicting evidence in the record.

Id.

As the Court noted, even if there is some x-ray evidence that indicates that there are opacities that would satisfy the requirements of prong (A), if there is other x-ray evidence available, or other evidence relevant to an analysis under prongs (B) or (C), then all of the evidence must be considered to determine whether the evidence as a whole indicates a condition of such severity that it would produce opacities greater than one centimeter in diameter on an x-ray. The Court stated:

> Of course, if the x-ray evidence vividly displays opacities exceeding one centimeter, its probative force is not reduced because the evidence under some other prong is inconclusive or less vivid. Instead, the x-ray evidence can lose force **only if other evidence affirmatively shows** that the opacities are not there or are not what they seem to be, perhaps because of an intervening pathology, some technical problem with the equipment used, or incompetence of the reader.

Id. (emphasis added).

In this case, there is considerable x-ray evidence that there are opacities that would satisfy the requirements of prong (A), in the form of the interpretations by Dr. Alexander. However, there is also other x-ray evidence available, as well as other evidence relevant to an analysis under prong (C), and thus all of the evidence must be considered to determine whether the evidence as a whole indicates a condition of such severity that it would produce opacities greater than one centimeter in diameter on an x-ray.

The numerous narrative x-ray interpretations in the record all describe the presence of large densities or masses in Mr. Roland's lungs, which many of the physicians attributed to complicated pneumoconiosis or massive pulmonary fibrosis. None of these interpretations, however, address the question of whether these densities or masses would appear on x-ray as Category A, B, or C opacities.[6] While these narrative interpretations do not fall in any of the categories of evidence specifically set out in the statute, however, they certainly do not detract from a conclusion that there are large

[6] While it would seem obvious, even to a layperson, that the multiple conglomerate masses described by Mr. Roland's physicians would show up on x-ray as opacities of *at least* 1 cm., I have not made that assumption.

opacities on Mr. Roland's x-rays, as reported by Dr. Alexander, or that Mr. Roland's condition was of such severity that it would produce such large opacities on x-ray. These narrative interpretations certainly do not provide affirmative evidence that the large opacities identified by Dr. Alexander are due to a disease process other than pneumoconiosis.

There is also evidence in the record that is relevant to an analysis under prong (C), in the form of the interpretations of CT scans. Thus, Dr. Butty reviewed Mr. Roland's December 23, 2000 CT scan, finding several large masses, the largest measuring approximately 6.2 cm. in greatest diameter. Dr. Alexander, who also reviewed this CT scan, found numerous large masses, which he found to be completely characteristic of the conglomerate fibrotic masses produced by the progressive massive fibrosis of complicated pneumoconiosis.

A CT angiogram of Mr. Roland's chest performed during his February 24, 2001 hospital admission showed conglomerate masses, which Dr. Cappiello described as multiple conglomerate masses of pneumoconiosis. Dr. Alexander also reviewed this CT scan, finding numerous large opacities, the largest being a contiguous large opacity, with a vertical length of 7cm., and a width of 5 cm. He stated that the location and appearance of these masses were completely characteristic of the conglomerate fibrosis seen in complicated pneumoconiosis.

While none of these interpreting physicians stated whether these masses met the statutory criteria, that is, whether, when x-rayed, they would show as opacities greater than one centimeter, their findings are certainly consistent with, and support Dr. Alexander's designation of large opacities on his ILO forms. Certainly, they do not establish that the opacities are not there, or that they represent something other than pneumoconiosis.

The Fourth Circuit stated that "because prong (A) sets out an entirely objective scientific standard" –i.e. an opacity on an x-ray greater than one centimeter –x-ray evidence provides the benchmark for determining what under prong (B) is a "massive lesion" and what under prong (C) is an equivalent diagnostic result reached by other means. Thus, the "other means" are medical tests, such as CT scans, that will provide a diagnostic result that is equivalent to the objective finding of an opacity on x-ray greater than one centimeter.

The reports by Dr. Vasudevan and Dr. Rasmussen do not properly fall under any of the prongs of Section 718.304, as they did not independently review any x-ray or CT scan films. Nevertheless, these reports certainly do not detract from the force of Dr. Alexander's x-ray findings, and the findings on CT scans. Indeed, virtually every physician who evaluated Mr. Roland concluded that he had severe obstructive pulmonary disease, in the form of complicated pneumoconiosis or massive pulmonary fibrosis.

Dr. Scott, Dr. Scatarige, and Dr. Wheeler reviewed Mr. Roland's more recent x-rays and CT scans at the request of the Employer, and uniformly concluded that they did

not show pneumoconiosis, complicated or simple. Each of these physicians acknowledged the presence of multiple, large masses in Mr. Roland's lungs, but felt that they were due to various causes, including granulomatous disease, tuberculosis, histoplasmosis, metastatic cancer, Wegener's, or sarcoidosis.

I find that the Employer has not offered affirmative evidence sufficient to cause Dr. Alexander's interpretations to lose force. Instead, Employer has relied on x-ray and CT scan interpretations that acknowledge the presence of large masses or processes, but speculate that they are the result of a variety of conditions other than pneumoconiosis, without sufficient corroboration or evidentiary support. Thus, Dr. Scott thought that the large masses he noted were "probably": or "possibly" tuberculosis or granulomatous disease. He relied on the fact that there was no background of small rounded opacities to suggest pneumoconiosis. However, Dr. Alexander discussed this at length, explaining the development of conglomerate masses of pneumoconiosis, and how they overtake and obscure the rounded opacities of simple pneumoconiosis. Even setting aside the fact that there is not a shred of evidence in the record to support a finding of tuberculosis, I find that Dr. Scott's conclusions are not persuasive, and certainly not affirmative evidence either that the large opacities noted by Dr. Alexander, and confirmed by Mr. Roland's treating physicians, are not there, or that they are due to a process other than complicated pneumoconiosis. Certainly, they did not provide an explanation that could be considered affirmative evidence that would cause the evidence that meets the standard under prong (A) to lose force.

Dr. Scatarige thought the large masses he saw on x-ray "could be" metastasis, but there were no small round opacities to suggest pneumoconiosis. Similarly, he found no small round opacities on Mr. Roland's CT scan, diagnosing either metastases, granulomatous disease, or Wegener's. Again, setting aside the fact that there is no evidence to suggest tuberculosis or cancer (a diagnosis that was considered by Mr. Roland's treating physicians), I find that Dr. Scatarige's conclusions are speculative, and certainly not affirmative evidence either that the large opacities noted by Dr. Alexander, and confirmed by Mr. Roland's treating physicians, are not there, or that they are due to a process other than complicated pneumoconiosis. Certainly, they did not provide an explanation that could be considered affirmative evidence that would cause the evidence that meets the standard under prong (A) to lose force.

Finally, Dr. Wheeler, who is also a highly qualified physician, and a B-Reader and Board Certified Radiologist, provided a variety of possibilities, including cancer, tuberculosis, or sarcoid.[7] But even setting aside the speculative nature of Dr. Wheeler's conclusions, I find that considerable doubt is cast on Dr. Wheeler's opinions by his statement that, unless Mr. Roland worked as an unprotected driller during or before World War II, it was unlikely that he had "large opacities," is., complicated

[7] It is important to note that nothing in the record indicates that Claimant was ever diagnosed with, treated for, hospitalized for, complained of, or displayed symptoms of tuberculosis.

pneumoconiosis. This statement suggests that Dr. Wheeler believes that complicated pneumoconiosis is a thing of the past, and that industrial hygiene has progressed to the point that it is no longer possible, or at least very unusual, for a miner to contract advanced coal workers' pneumoconiosis. He offers not a shred of medical or other scientific support for this statement.

I find that these interpretations do not constitute affirmative evidence that the large opacities as reported by Dr. Alexander are not there, nor do they establish that these masses are the result of a disease process other than pneumoconiosis. I find that these interpretations are speculative, especially in the absence of any evidence of tuberculosis, and that their reliance on the lack of a background of small opacities, which was persuasively addressed by Dr. Alexander, is misplaced.

Thus, I find that the Claimant has established by a preponderance of the credible and reliable evidence that Mr. Roland had complicated coal workers' pneumoconiosis. As such, she is entitled to the irrebuttable presumption that Mr. Roland's death was due to pneumoconiosis.

But even if I were to find that the Claimant was not entitled to the irrebuttable presumption that Mr. Roland's death was due to pneumoconiosis, I also find that the medical opinion evidence is more than sufficient to establish that pneumoconiosis caused, contributed to, or hastened his death. I note that virtually all of the reports regarding Mr. Roland's death attribute his demise to complicated pneumoconiosis, or respiratory failure due to pneumoconiosis. Thus, Dr. Vasudevan, Mr. Roland's treating physician, stated in his December 21, 2004 letter that Mr. Roland died of chronic respiratory failure and acute exacerbation of chronic obstructive pulmonary disease, caused by his complicated coal workers' pneumoconiosis/progressive massive fibrosis. (CX 4, DX 20). According to Dr. Vasudevan, complicated pneumoconiosis was Mr. Roland's primary condition, causing progressive lung impairment and ultimately his death. This opinion is consistent with and supported by the hospital records, which document a severe and worsening respiratory condition, reflected by large masses of complicated pneumoconiosis.

Dr. Rasmussen concluded that Mr. Roland's death was due primarily to his complicated coal workers' pneumoconiosis, and indeed that his classical radiographic features over time, with his progressively severe impairment in lung function, virtually excluded any other cause for his death other than pneumoconiosis. Dr. Vasudevan, who treated Mr. Roland, also concluded that his death was the consequence of his complicated pneumoconiosis.

On the other hand, seizing on a notation on the death certificate that "headache" was a significant condition contributing to Mr. Roland's death, Dr. Crisalli concluded that his death was "more related to whatever was causing his headache and not related to his chronic respiratory disease." Remarkably, Dr. Crisalli stated that there was nothing in the medical records to indicate that Mr. Roland died as a result of his pulmonary disease. Of course, the same death certificate that lists "headache" as a contributing condition in Mr. Roland's death states that Mr. Roland died as a direct result of cardiopulmonary

failure, as a consequence of complicated coalminer's lung. (DX 10). Nor did Dr. Crisalli address the records and report of Dr. Vasudevan, who stated unequivocally that Mr. Roland's death was due to his severe and progressive complicated pneumoconiosis.

I find that Dr. Crisalli's speculation that some sort of undiscovered and undiagnosed intracerebral event caused Mr. Roland's death, in the face of overwhelming evidence that Mr. Roland suffered from severe complicated pneumoconiosis and died a respiratory death, is entitled to absolutely no weight.[8] As Dr. Rasmussen noted, there are multiple possible immediate causes of Mr. Roland's death, but many of them, including central nervous system diseases, represent a combination of respiratory failure with a superimposed event. Although whatever caused Mr. Roland's headache cannot be determined, there is ample evidence of a progressively severe lung disease that was capable of causing death at any moment, especially during his last hospitalization.

I find the conclusions of Dr. Vasudevan, Dr. Rasmussen, and Dr. Alexander to be eminently well reasoned and amply supported by the objective medical evidence of record. Indeed, as with my finding regarding the existence of complicated pneumoconiosis, I find the medical evidence to be overwhelming in establishing that Mr. Roland died as the result of his pneumoconiosis.

CONCLUSION

I find that the Claimant has established by a preponderance of the medical evidence that Mr. Roland's death was caused by, contributed to, or hastened by pneumoconiosis. She is therefore entitled to benefits under the Act.

ORDER

Based on the foregoing, IT IS HEREBY ORDERED that the claim of Marjorie M. Roland, surviving spouse of Boyd M. Roland, for black lung benefits under the Act is hereby **GRANTED.**

IT IS FURTHER ORDERED that the Employer, Eastern Associated Coal Corporation, shall pay to the Claimant all benefits to which she is entitled under the Act.

SO ORDERED.

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[8] I also note that Dr. Crisalli utterly failed to address the question of whether Mr. Roland's pneumoconiosis contributed to or hastened his death, whether or not it was the primary cause of that death.

LINDA S. CHAPMAN
Administrative Law Judge

ATTORNEY'S FEES

An application by Claimant's attorney for approval of a fee has not been received. Thirty days is hereby allowed to Claimant's counsel for submission of such an application. A service sheet showing that service has been made upon all the parties, including the claimant, must accompany the application. The parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).